# Predictive toxicogenomics for the identification of chemical carcinogens : application to human hepatic cell lines

**Propositions**
belonging to the dissertation:

# Predictive toxicogenomics for the identification of chemical carcinogens:
## *Application to human hepatic cell lines*

*by*
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All models are approximations. Essentially, all models are wrong, but some are useful. However, the approximate nature of the model must always be borne in mind.

*G.E.P. Box, N.R. Draper*

Gene expression levels of HepaRG cells closely resemble those observed in human hepatocytes and human liver *in vitro*.

*This thesis - Chapter 2*

For the identification of genotoxic carcinogens, gene expression profiles induced in HepG2 cells by chemical agents perform better than those induced in HepaRG cells.

*This thesis - Chapter 2*

Evaluation of gene expression alterations induced in HepG2 cells by chemical agents facilitates the identification of false positive results from standard *in vitro* genotoxicity assays.

*This thesis - Chapter 3*

Transcriptomics approaches will overlook essential information, unless all possible functions of all known genes are fully elucidated.

*This thesis - Chapter 5*

Going from a whole genome to just a few sentinel genes is proof that the circle [of the microarrays era] is closing.

*J.M. Garcia-Sagredo*

The combination of Ames bacterial mutagenicity assay with specific gene expression alterations in HepG2 cells provides a novel and highly accurate *in vitro* genotoxicity test battery.

*This thesis - Chapter 4*

Given a large mass of data, we can by judicious selection construct perfectly plausible unassailable theories – all of which, some of which, or none of which may be right.

*P.A. Srere*

It is a capital mistake to theorize before one has data. Insensibly one begins to twist facts to suit theories, instead of theories to suit facts.

*A.C. Doyle*